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The role of AmtB, GlnK and glutamine synthetase in regulation of transcription factor tnra in bacillus subtilis

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Abstract

The nitrogen is a macroelement for all alive cells, from bacteria to animals. Although NH3/NH4 are highly toxic to animal, they are the preferred source of nitrogen for the most microorganisms and are assimilated by glutamine synthetase in the GOGAT cycle. The nitrogen limitation triggers a number of regulatory processes and activates many genes, providing the utilizing of alternative nitrogen sources. In Bacillus subtilis the genes of nitrogen metabolism are regulated by the transcription factor TnrA. In a cells it is bound to AmtB-GlnK proteins, the interaction with Glutamine synthetase (GS) represses its DNA-binding activity. Here we show the lack of AmtB leads to the nitrogen deficiency in a cell and, consequently, the increased expression of TnrA-de-pendent genes. In the lack of GlnK the transcription factor TnrA is constitutive bound to GS, the TnrA activity is repressed even under nitrogen limit conditions. Apparently, the TnrA activity is subjected to permanent repression by GS. In the absence of GS, the TnrA activity is strongly higher in compare to control, even under nitrogen limitation, when GS is active. These data allow to suggest that TnrA activity is regulated by the competitive binding to GlnK and GS.

Keywords

Bacillus subtilis, Glutamine synthetase, Nitrogen metabolism, Transcription factor TnrA